BEFORE THE APPEALS BOARD
FOR THE
KANSAS DIVISION OF WORKERS COMPENSATION

MITCHEL R. MERCIER	)	
Claimant	)	
VS.	)	
	)	Docket No. 258,302
BRADLEY REAL ESTATE	)	
Respondent	)	
AND	)	
	)	
RELIANCE NATIONAL INSURANCE COMPANY	)	
Insurance Carrier	)	

ORDER

Claimant appeals the March 16, 2009, Award of Administrative Law Judge Bruce E. Moore (ALJ). Claimant was awarded benefits for a 50 percent impairment of function to each lower extremity at the level of the lower leg, as the result of leg injuries suffered on August 3, 1999.

Claimant appeared by his attorney, Jeffrey E. King of Salina, Kansas. Respondent and its insurance carrier appeared by their attorney, Douglas C. Hobbs of Wichita, Kansas.

The Appeals Board (Board) has considered the record and adopts the stipulations contained in the Award of the ALJ. The Board heard oral argument on August 5, 2009.

ISSUES

1. Respondent contends claimant's disability is limited to his lower extremities, at the level of the lower leg. Claimant contends that his hypercoagulable state was caused or aggravated by the accident on August 3, 1999, or was made worse by the authorized treatment by board certified family practitioner Kevin D. Norris, M.D. Therefore, the development of the Budd-Chiari syndrome and subsequent liver problems are causally related to the work accident and claimant is entitled to an award for a permanent partial whole body disability. What is the nature and extent of claimant's disability?

2. Did claimant's Budd-Chiari syndrome arise out of and in the course of his employment with respondent? If so, is claimant entitled to past and future medical treatment for the hypercoagulable state, the development of Budd-Chiari syndrome and the need for ongoing, lifelong treatment with Coumadin?

FINDINGS OF FACT

Claimant had been employed as a maintenance supervisor for respondent for about four years, when, on August 3, 1999, he was running a machine that used wax stripper. The chemical being used on that date was called Square One, which contains sodium hydroxide and butoxyethanol. It is an industrial strength stripper. However, respondent had no MSDS sheets on that particular chemical. As claimant was running the machine, some of the stripper was splashing onto his legs. Claimant had no safety clothing and had not been told to wear safety clothing. After claimant went home that night, his legs began to swell and started to discolor. Claimant contacted a cousin, who is a nurse and works for Dr. Norris. Claimant was scheduled for an appointment the next day to see Dr. Norris. Claimant was diagnosed with a blood clot and cellulitis and, as a result, developed deep vein thrombosis (DVT). Initially, Dr. Norris took claimant off work for a couple of weeks. Claimant was treated with antibiotics and compression. After ultrasound testing on September 28, 1999, confirmed the DVT, claimant was started on Coumadin, a blood thinner. Claimant returned to work after two weeks and worked continuously until November 1999, when his job was eliminated. Dr. Norris determined that there was a cause-and-effect relationship between the chemical burns from the stripper and the subsequent cellulitis and swelling in the legs and formation of the DVT. The initial plan was to have claimant on Coumadin for four to six months. In November 1999, claimant was discovered to have anticardiolipin antibodies which placed him in a hypercoagulable state. In approximately May 2000, claimant was taken off Coumadin for a short period of time.

In July 2000, claimant was experiencing abdominal pain and ongoing lower extremity swelling. An ultrasound revealed ascites, heterogeneous liver and splenomegaly. Ascites is intra-abdominal fluid which causes abdominal pain. Heterogeneous liver is an abdominal condition of the liver revealed by ultrasound wherein one part of the liver appears different from another, indicating excess congestion and blood in different parts of the liver. Splenomegaly is the enlargement of the spleen caused from blood being backed up into the spleen. Tests performed at this time indicated elevated liver function, but a hepatitis series was obtained and was negative. Claimant was advised to undergo a liver biopsy, but did not at that time.

In October 2000, further testing revealed massive ascites. In November 2000, claimant had an upper gastrointestinal hemorrhage. A liver biopsy showed extensive necrosis of centrilobular hepatocytes and an accumulation of blood consistent with hepatic venous outflow obstruction. At this point, claimant was diagnosed with Budd-Chiari

syndrome, a condition where blood clots plug up the hepatic veins which drain the liver. As a result, claimant's liver began to fail. He has been told that he will ultimately need a liver transplant. In order to delay what is apparently inevitable, claimant had a transjugular intrahepatic portosystemic shunt (TIPS) procedure done to decompress the liver and inhibit clots from forming.

Following the filing of a workers compensation claim for injuries arising out of his chemical exposure, a preliminary hearing was held on April 13, 2004. At that time, the parties appeared and a conference was held with the ALJ. Although no formal transcript was taken, it appears from the parties' briefs that both claimant and respondent had obtained written medical opinions relating to the causation of claimant's liver disease. Claimant offered the opinion of Jameson Forster, M.D., who attributed claimant's Budd-Chiari syndrome to his occupational exposure to chemicals in August 1999, while respondent offered the opinion of Allen J. Parmet, M.D., who disputed that claimant's occupational exposure was the cause of his present diagnosis.

When presented with this evidence, the ALJ appointed Mark D. Uhl, M.D., to perform an independent medical examination pursuant to K.S.A. 44-516. Dr. Uhl is a gastroenterologist and devotes approximately 15 percent of his practice to those with liver disease. While he has treated as many as 15 patients with Budd-Chiari syndrome, it is a very uncommon liver disease and he has never encountered a case where Budd-Chiari syndrome was caused by chemical exposure.[1]

Following an examination in June 2004, Dr. Uhl authored a report dated July 25, 2004. In both his report and during his deposition, Dr. Uhl indicated claimant is suffering from a hypercoagulable state that had given rise to Budd-Chiari syndrome. Simply put, when drainage of the liver is blocked, here by virtue of claimant's hypercoagulated blood, the blood will back up into the liver, causing it to swell and impair its ability to function. The TIPS procedure can re-route the blood through a shunt and will "buy the patient some time," but a liver transplant will eventually become necessary

Dr. Uhl performed computerized medical research on Medline, the national data bank for medical research. Dr. Uhl found no connection in any of the medical literature between cutaneous exposure to 2-butoxyethanol and sodium hydroxide and Budd-Chiari syndrome. Thus, based upon his independent research and his own professional experience, Dr. Uhl expressed the belief that claimant developed a hypercoagulable state, but does not know what caused claimant's hypercoagulable state.[2] Dr. Uhl agrees that it is possible that chemicals might have affected claimant's protein levels such that they

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[1] Uhl Depo. at 28 and 31-32.

[2] *Id.* at 22.

caused claimant to develop his hypercoagulable state.[3] However, he went on to opine that it was his understanding that "people can have a hypercoagulable state without any predisposing event that caused a hypercoagulable state. A lot of people I think probably have a hypercoagulable state and have not yet had their clot."

On December 8, 2004, claimant filed an Application for Preliminary Hearing, claiming he was being treated for his DVT with Coumadin and requesting that respondent be ordered to pay for his Coumadin prescriptions. After a hearing on January 12, 2005, the ALJ authorized Dr. Norris to treat claimant solely for purposes of his DVT.

On September 27, 2005, claimant filed another Application for Preliminary Hearing, requesting treatment for his liver condition. He claimed that Dr. Parmet had issued a report indicating that his Budd-Chiari syndrome developed as a result of treatment or discontinuation of treatment from the work-related injury. At a hearing on January 13, 2006, claimant introduced, among other records, a report of Dr. Parmet dated August 29, 2005. In that report, Dr. Parmet stated that once claimant was identified as having a hypercoagulable state, he should not have had his Coumadin stopped, and then stated: "He did not have Budd-Chiari syndrome prior to cessation of the anticoagulants, but did develop it after that time."[4] In an Order dated January 13, 2006, the ALJ found that claimant was entitled to medical care for his end-stage liver disease and his DVT. Dr. Norris remained designated as claimant's treating physician for his DVT. Respondent was to provide claimant with the names of three qualified physicians from which claimant could choose one as an authorized treating physician for his end-stage liver disease.

Dr. Parmet's deposition was taken by respondent on March 15, 2006. Dr. Parmet testified that as a result of being contaminated with cleaning chemicals, claimant suffered chemical burns to his legs. Claimant developed cellulitis and subsequently developed a DVT in his left leg. The DVT was caused by the burn injuries, but was also partially caused by claimant's hypercoagulable state, which was a preexisting, genetic problem caused by a lack of a protein that prevents clots from forming. Dr. Parmet stated that claimant could have developed the clot without having had the burns, but in this setting, he opined that the burns contributed to the clots in claimant's leg. Claimant was treated with the anticoagulant Coumadin for six months after the formation of the DVT, which is the normal protocol, unless the hypercoagulable state is present. Once a hypercoagulable state is identified, a person should be anticoagulated for life. When claimant's Coumadin was stopped, this led to the formation of a clot in the portal system, causing damage to claimant's liver and the development of the Budd-Chiari syndrome.

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[3] *Id.* at 25.

[4] Parmet Depo., Ex. 3 at 2.

Dr. Parmet opined that when claimant's Coumadin treatment ended in the spring of 2000, his condition concerning his legs and DVT would have been at maximum medical improvement.

In the fall of 2000, claimant suffered another clot, this time in the portal system. This second clot damaged claimant's liver and caused his Budd-Chiari syndrome. The portal system is isolated from the other veins in the body and flows to the liver from the gastrointestinal tract. Consequently, Dr. Parmet opined that there is no mechanical connection between the DVT in claimant's leg and the blood clotting in his liver. The vein in claimant's leg where the DVT formed is unrelated to the portal system.

Dr. Parmet stated that because of claimant's hypercoagulable state, he should have been continued on Coumadin rather than having it stopped after six months. Although claimant's lifelong need for anticoagulation was not caused by the chemical burns or the DVT, the burns and the development of the DVT should have served as notice to look for and identify a hypercoagulable state. The burns and development of the DVT in claimant's leg did not cause or necessitate lifelong anticoagulation. The need for lifelong anticoagulation is based solely upon claimant's preexisting hypercoagulable state. Claimant's hypercoagulable state existed before his chemical burns and remained unchanged thereafter. Having a DVT in his leg did not change his coagulation capability. Dr. Parmet stated that claimant's Budd-Chiari syndrome is not related to the August 1999 chemical exposure. Claimant's preexisting hypercoagulable state led to the portal vein thrombosis and subsequently led to his Budd-Chiari syndrome.

Claimant was referred by his attorney to board certified occupational medicine, emergency medicine and preventative medicine specialist P. Brent Koprivica, M.D., for an examination on January 19, 2008. Dr. Koprivica diagnosed claimant with chemical exposure and resulting chemical burns on both legs. As a complication of the chemical burns, claimant developed DVT in his left lower extremity and was treated with anticoagulants. Dr. Koprivica determined that claimant was already hypercoagulable at the time of the accident. But, the DVT aggravated or intensified the underlying hypercoagulable condition. And, he opined that the DVT developed from the exposure to the chemical and the development of the burns on claimant's legs. Pursuant to the fourth edition of the AMA *Guides*,[5] Dr. Koprivica rated claimant at 50 percent to each lower extremity at the level of the lower leg. Claimant was also rated at 40 percent to the whole body for the liver damage. The ratings combine to a 62 percent whole person impairment.

On cross-examination, Dr. Koprivica acknowledged that, in his opinion, claimant's hypercoagulable state was a preexisting condition. It is a genetic condition, and claimant has had it all his life. He also agreed that the blood clot in claimant's leg did not travel to claimant's liver as they cannot be physically connected. However, due to the

[5] American Medical Association, *Guides to the Evaluation of Permanent Impairment* (4th ed.).

hypercoagulable state, claimant needs to be on anticoagulation medication, like Coumadin, for the rest of his life.

The deposition of Dr. Parmet was again taken on August 20, 2008. Dr. Parmet had done additional research into the relationship between DVT and Budd-Chiari syndrome. He reviewed an article by Paolo Prandoni, M.D., Ph.D., et al., titled "The Long-Term Clinical Course of Acute Deep Venous Thrombosis" as well as other articles on the DVT subject. The study questioned people with DVT, and the risk of developing a recurrent clot. The predisposition risk was 40 percent. The group study found no reports of episodes of Budd-Chiari syndrome. Nothing in the scientific literature connected DVT in the periphery with Budd-Chiari syndrome. He acknowledged that claimant had an increased risk of re-clotting in the extremities. But, there was no connection with the risk of developing a clot in the hepatic vein draining the liver. Claimant's hypercoagulable state, which contributed to his DVT in 1999, was the sole cause of his Budd-Chiari syndrome in 2000. Dr. Parmet also acknowledged that the propensity to clot, which claimant had, could cause DVT of the periphery and could also cause Budd-Chiari syndrome, which he described as basically DVT of the hepatic vein. He agreed that the proper course of treatment would be to give claimant an anticoagulant for life.

PRINCIPLES OF LAW AND ANALYSIS

In workers compensation litigation, it is the claimant's burden to prove his or her entitlement to benefits by a preponderance of the credible evidence.[6]

The burden of proof means the burden of a party to persuade the trier of fact by a preponderance of the credible evidence that such party's position on an issue is more probably true than not true on the basis of the whole record.[7]

> If in any employment to which the workers compensation act applies, personal injury by accident arising out of and in the course of employment is caused to an employee, the employer shall be liable to pay compensation to the employee in accordance with the provisions of the workers compensation act.[8]

When a primary injury under the Workers Compensation Act arises out of and in the course of a worker's employment, every natural consequence that flows from that injury is compensable if it is a direct and natural result of the primary injury.[9]

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[6] K.S.A. 1999 Supp. 44-501 and K.S.A. 1999 Supp. 44-508(g).

[7] *In re Estate of Robinson*, 236 Kan. 431, 690 P.2d 1383 (1984).

[8] K.S.A. 1999 Supp. 44-501(a).

[9] *Gillig v. Cities Service Gas Co.*, 222 Kan. 369, 564 P.2d 548 (1977).

Although this record does not support a direct connection between claimant's chemical exposure on August 3, 1999, and the later development of Budd-Chiari syndrome, the record does support a connection between the accident and the development of the DVT. And, the treatment claimant was provided for the DVT contributed to claimant's liver damage. Claimant was placed on the Coumadin as treatment for the DVT. Additionally, once the hypercoagulable state was diagnosed, the general medical consensus is that claimant should have remained on the anticoagulation medication for life. Dr. Norris began treating claimant in 1999 with Coumadin for the DVT. While claimant was on the Coumadin, he was found to be in a hypercoagulable state. It is generally agreed by the medical experts in this record that once a person is diagnosed in a hypercoagulable state, the administration of the anticoagulation medication was to continue for life. The failure to continue the Coumadin treatment resulted in the development of Budd-Chiari syndrome and caused or contributed to the liver damage.

The Kansas Supreme Court, in *Roberts*,[10] determined that where an injury is compensable under the Workers Compensation Act, any aggravation of that injury or any additional injury arising from the treatment of that injury is a consequence of the primary injury and becomes compensable.

Here, claimant developed DVT from the chemical exposure to sodium hydroxide and butoxyethanol. The appropriate treatment was to place claimant on an anticoagulant, here, Coumadin. To this point, the treatment was proper. However, in November 1999, as the result of edema, claimant developed DVT in the left lower extremity. That is the only extremity that had been diagnosed with DVT. At that point, claimant was discovered to have anticardiolipin antibodies which placed him in a hypercoagulable state, and he was started on Coumadin.[11] This diagnosis should have alerted the treating health care provider that claimant should remain on the anticoagulant for life. Instead, Dr. Norris took claimant off the Coumadin for a short period of time in 2000. It was at this point that the Budd-Chiari syndrome developed and the long-term liver damage occurred. Following the findings of *Roberts*, the Board finds that respondent and its insurance company are liable for the development of Budd-Chiari syndrome and the need for long-term treatment for claimant's liver as the result. This includes lifelong use of Coumadin or another anticoagulant and the need for a liver transplant in the future if necessary.

Pursuant to the opinion of Dr. Koprivica, claimant has suffered a 62 percent whole person disability.

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[10] *Roberts v. Krupka*, 246 Kan. 433, 790 P.2d 422 (1990).

[11] Norris Depo. at 51-52.

CONCLUSIONS

Having reviewed the entire evidentiary file contained herein, the Board finds the Award of the ALJ should be modified to award claimant a 62 percent functional disability to the whole body for the injuries and subsequent liver damage from the August 3, 1999, accidental injury and resulting treatment.

AWARD

WHEREFORE, it is the finding, decision, and order of the Appeals Board that the Award of Administrative Law Judge Bruce E. Moore dated March 16, 2009, should be, and is hereby, modified to award claimant a 62 percent whole person permanent partial disability.

WHEREFORE, AN AWARD OF COMPENSATION IS HEREBY MADE IN ACCORDANCE WITH THE ABOVE FINDINGS IN FAVOR of the claimant, Mitchel R. Mercier, and against the respondent, Bradley Real Estate, and its insurance carrier, Reliance National Insurance Company, for an accidental injury which occurred on August 3, 1999, and based upon an average weekly wage of $700.00.

Claimant is entitled to 148.71 weeks of temporary total disability compensation at the rate of $383.00 per week or $56,955.93, followed by permanent partial disability compensation at the rate of $383.00 per week for a 62 percent permanent partial general disability, making a total award not to exceed $100,000.00.

As of the date of this Order, the total award is due and owing and ordered paid in one lump sum less any amounts previously paid.

Although the ALJ's Award approves claimant's contract of employment with his attorney, the record does not contain a filed fee agreement between claimant and claimant's attorney. K.S.A. 44-536(b) mandates that the written contract between the employee and the attorney be filed with the Director for review and approval. Should claimant's counsel desire a fee be approved in this matter, he must file and submit his written contract with claimant to the Director for approval.[12]

[12] K.S.A. 44-536(b).

IT IS SO ORDERED.

Dated this ____ day of September, 2009.

BOARD MEMBER

BOARD MEMBER

BOARD MEMBER

c: Jeffrey E. King, Attorney for Claimant
Douglas C. Hobbs, Attorney for Respondent and its Insurance Carrier
Bruce E. Moore, Administrative Law Judge